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Reply to Mitteroecker and Fischer: developmental solutions to the obstetrical dilemma are not Gouldian spandrels

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In our study on human pelvic development (1) we proposed two hypotheses explaining the narrowing of the female pelvis during postreproductive life: Hypothesis 1 proposes ultimate, evolutionary causes, reflecting selective pressures acting on postreproductive life. Hypothesis 2 proposes proximate, developmental causes, reflecting reduced estrogen levels during postreproductive life.

Mitteroecker and Fischer (2) largely replicate our results with an independent data set, taking into account age effects on pelvic shape variation not considered in their earlier study (3) (note that the smoothing functions used in our study only serve for visual guidance, not for statistical testing). Rather than testing our hypotheses with new data, however, they discard the first hypothesis, and interpret postreproductive pelvic narrowing as an evolutionary side-effect. Here we reiterate that further research, and new empirical data, are required to actually test both hypotheses, before any of them can be falsified.

First, Mitteroecker and Fischer (2) argue that because postreproductive mothers had a small inclusive fitness, and were infrequent during human evolution, their pelvic phenotype was fitness-irrelevant. This argument is not valid. A long postreproductive lifespan is generally acknowledged to be one of the hallmarks of modern human evolution (4). Longevity likely results from positive selection on a large number of postreproductive physiological and morphological features, however small the inclusive fitness of the initially few postreproductive individuals may have been, and however small the contribution of each single feature to the “longevity phenotype” as a whole may be.

Second, the concept of biological spandrels (5) is helpful to categorize phenotypic characters into main products and by-products of natural selection, thus cautioning against adaptationist over-interpretation of single phenotypic features. However, this presupposes an omniscient observer who knows which structure has been selected for which function. In the case of the human pelvis, we are doubly blind: Since the falsification of Washburn’s obstetrical dilemma hypothesis (6-8), we do not currently understand which functional constraints (in addition to obstetric constraints) govern the divergent evolution of the male and the female pelvis. Also, we do not understand yet which mechanisms of sex-biased autosomal gene expression govern human pelvic development.

Third, we did not propose that postreproductive narrowing evolved specifically “as an adaptation to changing obstetric needs” (2). Rather, we proposed that it evolved under positive selection for long postreproductive lives (1). Based on the currently available data we proposed that humans evolved a highly effective developmental mechanism that governs female pelvic shape

change via changes in estrogen levels (which themselves represent a physiological marker of fertility). According to this hypothesis, both the pubertal widening and postmenopausal narrowing of the female pelvis are based on the same evolved mechanism of hormone-mediated developmental plasticity. Designating pelvic widening as the evolutionary main effect and narrowing as a side effect is premature unless we understand the fitness functions of pelvic morphology in both sexes, and how they change during an individual's lifetime.

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